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NIFEDIPINE INDUCED GINGIVAL ENLARGEMENT

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ABSTRACT

Gingival enlargements are most undesirable consequences of few drugs like anti-convulsants, immunosuppresants and calcium channel blockers. Gingival Enlargement (also known as Gingival hypertrophy or Gingival Hyperplasia) is an abnormal overgrowth of gingival tissues. Nifedipine is one of the commonest antihypertensive drugs (calcium channel blockers) used to treat hypertension and some forms of angina. Though it has various side effects, gingival enlargement is one of the most attended side effects by the dental practitioners. Nifedipine induced gingival enlargement is noticed in almost 10% of patients. It can be localized or generalized and can range from mild to severe, affecting patients appearance and function. Many cases will progress for long periods of time and it may be affected by secondary infection due to improper oral hygiene maintenance. This paper describes clinical features and management of gingival enlargement associated with nifedipine. Treatment generally employs substitution of the drug and then providing corrective surgery. Proper plaque control and effective oral hygiene can reduce its severity or prevent its occurrence.

KEYWORDS: drug induced, gingival enlargement, nifedipine.

INTRODUCTION:

The enlargement of gingiva is associated with more than 20 drugs used for various diseases [1]. Calcium channel blockers (CCBs) are the most commonly prescribed anti-hypertensive drugs for patients with cardiovascular disorders. Though it has various side effects, gingival enlargement is the most attended side effect which is of great importance in dentistry. Gingival hyperplasia on long term use of Nifedipine is rare in the literature [2]. The first documented case of Nifedipine induced gingival enlargement was reported in 1984 [3]. Advantages of nifedipine include its effectiveness, low cost and frequency of administration. Significant correlations between the occurrence and severity of nifedipine-induced gingival enlargement and the presence of plaque and calculus accumulation have been demonstrated [4]. These irritants stimulate the formation of granulation tissue that consists of proliferating endothelial cells, chronic inflammatory cells and few fibroblasts. The overzealous reaction to irritants manifests in inflammation and enlargement of gingiva causing functional disturbances and hinder. Moreover age and gender have also been considered as risk factors for drug induced gingival enlargement [5]. When the gingival tissue is fibrotic, resolution of enlargement may not occur, resulting in the persistence of periodontal pocketing such that effective oral hygiene is impeded. This scenario requires a more detailed assessment and management plan designed to map the extent of gingival and possibly periodontal involvement. Various systemic factors, including endocrinopathies, hematologic conditions, diet, and drugs, can modify the immune-inflammatory response [2]. The enlargement is generally painless unless traumatized during toothbrushing, flossing or eating. Role of genetic factors in these gingival lesions also has been investigated. Nonetheless, not all individuals who take these medications will develop enlargements of the gingival tissues, suggesting a susceptibility requiring specific characteristics. Finally, all of these drugs produce clinical lesions and histologic characteristics that are indistinguishable from one another. Its management involves a non-surgical and surgical approach. Non-surgical therapy involving scaling and root planing aims to remove local irritants to reverse the inflammatory changes and control of the etiologic factors. Splinting of mobile teeth helps in stabilizing dentition and providing optimum conditions for healing. When there is no significant reduction of enlargement, surgical approach like gingivectomy (External bevel or Internal bevel Gingivectomy) helps in restoring the gingival contours. Periodontal therapy is diagnosis- driven, it should include modification of risk factors which potentiates progression of plaque induced periodontal disease. In this article we have reported a case of gingival enlargement in a 60 year old hypertensive patient who was on low dose Nifedipine therapy.

CASE REPORT:

A 60-year old male patient with low socioeconomic status reported with chief complaint of swollen gums and difficulty in brushing for the past 1 month. He was a known hypertensive and was on medication for the same since 7 years (10 mg Nifedipine/day). On intraoral examination- pink, firm, irregular, nodular, non- tender enlargement involved the buccal aspect of marginal and attached gingiva of the mandibular arch. The gingiva was swollen, shiny, reddish pink in color **(Figure-1)**. The enlargements were asymptomatic in nature with no history of bleeding. Pseudopockets were present. History revealed that the swelling started progressively and attained the present size since 3 months. Therefore, considering the dental history and radiographs, gingivectomy was planned with slight gingivoplasty. The surgical technique was explained to the patient and informed consent was obtained. Patient's complete blood count, bleeding time, clotting time and platelet count were with in normal limits. Preparation of the patient included scaling and root planing of the entire dentition; and oral hygiene instructions. Considering medical status of the patient, drug was not altered because, dose taken by the patient was low (10 mg/day). Local anesthesia devoid of vasoconstrictor was used to remove fibrous tissue. After local anesthesia, pocket depths were measured and marked with a pocket marker. The thick, fibrous gingival tissue was excised with an external bevel incision from the lower anterior teeth, following a scalloped pattern around the gingival margin, with a 15 number blade. This was followed by a second incision, into the intracrevicular sulcus. The incision was extended distally 1 to 2 teeth to blend into the gingival sulcus of the untreated teeth. The third incision is then placed interproximally to release the interdental papilla. Additional minor gingival recontouring was performed to establish symmetrical gingival margins. The excised tissue obtained was sent for histological investigations. After control of bleeding, periodontal dressing was placed on the operated site. Patient was given analgesics and antibiotics to control any post-operative infections. There was no post-operative complication and healing was satisfactory after 10 days. The patient was instructed to use soft tooth brush for mechanical plaque control in the surgical area and 0.2% chlorhexidine rinses twice daily. Supportive periodontal maintenance at 3 months was prescribed to maintain periodontal health and to re-evaluate this area. The patient was recalled and followed at a period of 3 months, 6 months and 1 year intervals. No recurrence of growth was observed during any of the recall visits. At 12 months recall, there was successful healing with no recurrence and probing depth was minimal **(Figure-2)**. Histological report revealed hyperplastic and acantholytic stratified squamous epithelium with elongated rete ridges extending into connective tissue which was fibro collagenous and showed focal areas of fibrosis. Infiltration of chronic inflammatory cells and congested blood vessels were seen which suggested of gingival hyperplasia.

DISCUSSION:

Gingival enlargement due to drugs which are administered to treat the systemic problems is of major concern which needs attention. Gingival overgrowth is characterized by the accumulation of extracellular matrix in gingival connective tissues, particularly collagenous components, with varying degrees of inflammation [6]. Histopathologically, nifedipine induced drug enlargement also reveals by accumulation of extracellular matrix and ground substance, with a parakeratinised epithelial layer and deep ridges penetrating into the underlying connective tissue. The esthetic rehabilitation of such patients involves a multidisciplinary approach. Periodontal surgery is generally perceived as excisional in nature with pocket elimination being the treatment goal. Gingivectomy is a procedure that is performed for reduction of excessive gingival overgrowth with a simultaneous consultation with a physician for drug dosage manipulation. However, long term use of these anti-hypertensive drugs causes gingival enlargement in few cases. In a community-based study, it was noticed that more than 6% of subjects taking Nifedipine had significant overgrowth and it was directly proportional to amount of gingival inflammation [7]. As suggested by Seymour et al. drug induced gingival hyperplasia is a

multifactorial disease. Few of the reasons for gingival enlargement in our case can be due to defective collagen activity or due to decreased uptake of folic acid, blockage of aldosterone synthesis from adrenal cortex and an increase in adreno corticotropic hormone (ACTH) level and up regulation of keratinocyte growth factor [8]. Drugs like Nifedipine, block influx of calcium ions thereby affecting homeostasis of collagen. Synthesis and degradation of collagen being altered leads to the abnormal growth. Evidence from animal studies confirms that, nifedipine when added to gingival fibroblast in culture, increase the conversion of testosterone to 5α dihydrotestosterone and this active metabolite would target subpopulations of fibroblasts [9]. Idiopathic/Hereditary gingival enlargement from our case was ruled out as these enlargements are commonly detected at an early age and in few cases even at birth. The dose of drug in present case was not altered as the dose was very low. Several studies in literature have suggested that a dose range of 30-60 mg/day is more associated with gingival enlargement. Dose of Nifedipine taken by patient was below the threshold limit of gingival overgrowth. Relation between gingival hyperplasia and pharmacokinetics of the drug has been investigated and are much debatable [10]. This threshold might differ from patient to patient which might not be a suitable prognostic factors for gingival enlargement. Every effort should be made to obtain primary closure of the surgical site to facilitate healing and so discourage proliferative granulation tissue formation which heralds early recurrence. Follow-up is required to ensure, if any recurrence is detected early and dealt with. The decrease in the size of the lesion is directly proportional to the amount of inflammation present. If the lesion is composed mostly of fibrous tissue, there is little shrinkage, but if considerable granulation tissue and inflammation exist, there is marked shrinkage. Management of such inflammatory enlargements require regular professional oral prophylaxis and good patient compliance.

CONCLUSION:

A thorough discussion and consideration of the various differential diagnoses should be done carefully to prevent unnecessary distress to the patient and family. Although nifedipine induced gingival enlargement is very less, there is a need for physicians and dentist to make a coordinated treatment plan and practice care while prescribing these drugs which are associated with gingival overgrowth.

Figure 1

Figure 2

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